

muscles, which are supplied by the ulnar nerve, was beautifully shown, as, although both flexor tendons were completely divided, the patient could still close the fingers, it is true in a loose sort of way, but a careless observer would probably have concluded that the flexor tendons had not been injured.

The ability to approximate the thumb to the fingers must be explained, it seems to me, on the ground that the adductors and part of the short flexor of the thumb, which are supplied by the ulnar nerve, act to some extent for the opponens pollicis, but that such substitution does not always take place is shown by the result in the second case reported.

CASE V.—Saw wound of the axilla dividing the axillary vessels and the median, ulnar, musculospiral, and internal cutaneous nerves; suture of the nerves; gangrene; amputation below the elbow.

A. S., a white male, aged eighteen years, was admitted to the Emergency Hospital July 7, 1907, having the day before been thrown against a circular saw. Examination showed a large ragged wound of the right axilla and right side of the chest, extending to, and grooving the neck of, the humerus and dividing the pectoralis major, the axillary vessels, and the median, ulnar, musculospiral, internal cutaneous, and lesser internal cutaneous nerves. The vessels had been closed by the formation of thrombi. The nerves were united after considerable difficulty in finding the ends and determining which ends belonged together, using silk and overlapping the ends.

The lower ends of the median and musculospiral nerves were concealed several inches below the wound, and dissection of the arm below the wound was necessary in order to find them. The wound was partially closed and drainage provided. Next day the hand was cool, and slight gangrene of the edge of the wound was noticed.

July 16, a line of demarcation had formed about the middle of the forearm, so that the forearm was amputated about three inches below the elbow. Recovery followed, but it is too early to expect any restoration of function in the muscles supplied by the sutured nerves.

THE PATHOGENESIS OF TABES DORSALIS.¹

BY TOM A. WILLIAMS, M.B., C.M. EDIN.,
OF WASHINGTON, D. C.

THE pathogenesis of tabes dorsalis has excited a great deal of discussion ever since Duchenne² first differentiated the locomotor

¹ Read by invitation at a meeting of the Philadelphia Neurological Society, February 24, 1908.

² Arch. de méd., 1858.

ataxia he so well describes. None of the more recent reviews of the subject has formulated very definite conclusions, nor acceded categorically to a single theory; none of the framers of these has quite succeeded in accounting for all the clinical facts already ascertained. Having had exceptional opportunities during the last two years to examine the preparations of many observers and to study a very large number of cases of *tabes fruste*, I wish in this presentation to indicate, as briefly as possible, the reasons for the conclusion reached that in the main the true explanation is that of Nageotte.³ An attempt is made also to account for the discrepancies between the findings of others and those of Nageotte's remarkable studies upon the radicular nerve.

Avoiding all arbitrary divisions, types, and stages of disease, and sketching as far as possible the processes which occur and the evolution of the symptoms, I postulate in the first place, with the great majority of neurologists, that the tabetic process differs not in kind, but only in locality from the paralytic process, and in the second place I reject the explanation, familiar in the text-book, that *tabes* is a primary dystrophy either of the posterior column or of the sensory protoneuron as a whole or an atrophy thereof, directly caused by alcohol, prolonged strain, syphilis, or sexual excesses. To make this clear a few anatomical and physiological details must be recalled.

THE LESIONS ON THE POSTERIOR ROOTS.—It will be recollected that in or near the row of foramina between the laminae of the vertebrae are situated the ganglia upon the posterior roots of the spinal cord, and that the lower ganglia are a considerable distance inside the spinal canal. The nutrition function of the ganglion cells is too well known to require emphasis. But it is perhaps insufficiently borne in mind that the regenerative function of these cells is efficacious only when the fiber concerned possesses a neurilemma, and that within the spinal cord the axons of the root nerves lose this sheath, retaining only the white substance of Schwann.

Between the lower ganglia and the segment of the spinal cord corresponding with each there intervenes several inches, which the radicular nerve traverses in contact with the leptomeninges, which arrangement is shown in Fig. 1. This shows clearly the canal formed by the pachymeningeal reflection as the nerve pierces the theca vertebralis to become peripheral. In this canal it is probable that communication takes place between the cerebro-spinal fluid filling it and the lymph in the sheath of the peripheral nerve (Orr and Rows⁴). Figs. 2, 3, 4, 5, and 6, show the lesions

³ Soc. de Biol., January, 1901; July, November, December, 1902; July, 1904; May, October, 1905; January, 1906. Note sur les formations cavitationnaires par périnévrine dans les nerfs radiculaires, *Rev. neur.*, 1907. Sur la systematisation dans les affections du système nerveux et en particulier dans le *tabes*, Congrès Neur. de Paris, 1900. *Neuv. Icon. Salpet.*, 1904, p. 1. *Pathogénie du Tabes Dorsalis*, Paris, 1903. *Rev. neur.*, 1906; January, 1903.

⁴ *Rev. Neur. and Psy.*, 1903, p. 639; 1906, p. 25; 1907, p. 346. *Brain*, 1904, p. 460.

described by Nageotte in this locality in cases of tabes and tabo-paralysis. Examination shows this lesion in all stages from simple

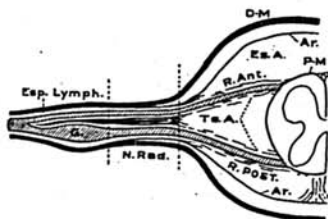


FIG. 1.—Diagram showing the relations of the meninges to the radicular nerve. *N.Rad.*, radicular nerve; *PM*, pia mater; *Ar.*, arachnoid; *DM*, dura mater; *R.Ant.*, anterior roots; *R.Post.*, posterior roots; *G.*, ganglion; *E.L.*, facultative lymphatic space. (From Nageotte.)

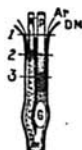


FIG. 2.—*AP*, anterior and posterior roots; *Ar.*, arachnoid; *DM*, dura mater; *G.*, intervertebral ganglion. (From Nageotte.)

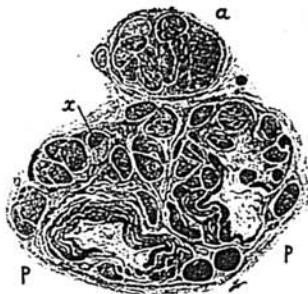


FIG. 3.—Cross-section of the radicular nerve in a general paralytic in which the lesions of the anterior root (*a*) were more prominent than those of the posterior root (*p*). The section is at line 3 of Fig. 2 in which the posterior root is involved also. Note the perineuritis with its cavity formation, and the paler bundles of degenerated fibres even where there is no perineuritis. The bundle *x* is half intact. (From Nageotte.)

round-cell infiltration, through granuloma, toward necrosis with cavity formation. They are the fruit of complete serial sections of several radicular nerves in each of eleven cases of this disease.

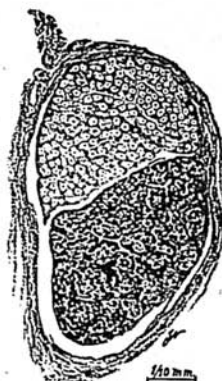


FIG. 4.—The region *x* in Fig. 3 magnified 90 diameters to show the healthy fibers and those from which the myelin has disappeared. (From Nageotte.)

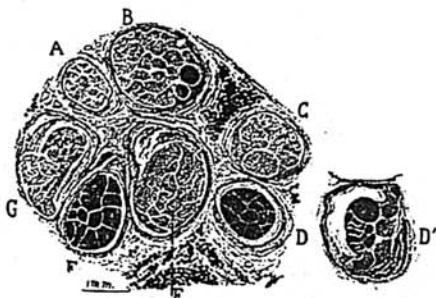


FIG. 5.—Cross-section of the radicular nerve in a tabetic, showing the round-cell infiltration. The bundles *F* and *D* are spared, while all the rest are degenerated. *D'* is a cross-section of *D* lower down. Notice the area of lacunar perineuritis which displaces some fasciculi. Lower still this necrosis was found to occupy the centre of the bundle. (From Nageotte.)

Now, even the chronicity of the infection and the comparatively indolent reaction of the granulomatous process are in this situation sufficient to affect seriously the nerve fibers contiguous to the in-

flammatory area; for in the circumscribed canal of unyielding dura mater, the pressure of toxic effects of the exudate impinges upon the nerve fibers surrounding. These are either destroyed, or at least sufficiently injured to prevent the passage of the impulses from the ganglion cell, which subserves the trophicity of the distal portion of the fiber, which accordingly degenerates. Their lack of neurilemma prevents their regeneration, even after the granuloma has undergone absorption; and the postmortem appearance of tabes dorsalis is thus constituted. Regeneration, however, does occur in the posterior root itself just as far as Obersteiner's ring; and it is to this cause that we must attribute the many attempts to refer the tabetic process to a primary dystrophy; for the earlier pathologists had not conceived that what appeared a healthy structure, was the real source of the lesion which they found in the posterior column of the spinal cord.

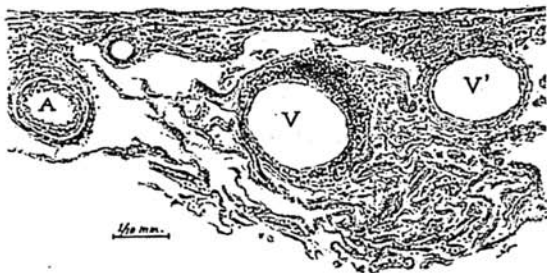


FIG. 6.—An area of old meningitis in a tabetic. The process is in resolution except around *V*, where an inflammatory nodule is in full development. Note the sclerotic atrophy of the walls of the artery *A* and veins *V* and *V'*. (From Nageotte.)

Nageotte's opinion that the process is toxic does not accord with the findings of similar posterior column degenerations with evidence of pressure upon the roots, in cases of cerebral tumor (Lejonne*) in which the manometric pressure of the spinal fluid is increased; and it would seem that the mechanical factor of the granulomatous infiltration was enough to account for the tabes, although the experiments of Orr and Rows show that causes purely toxic may produce degenerative changes.

THE DISPARATE DISTRIBUTION OF THE LESIONS.—The preponderance of the process upon the fibers of deep sensibility is connoted by the name given to the disease by Duchenne, of Boulogne, locomotor ataxia. Its occurrence may be due to as yet unascertained peculiarities in the course of these fibers, which we

* *L'Encéphale*, March, 1907.

know to accompany the motor nerves. A search among carefully described cases of tabes, however, shows that other anomalies of sensation are very much commoner than the classics allow; it is probable, therefore, that it is merely the inconvenience entailed by the impairment of this function which is the cause of the stress laid upon its involvement when the pathology of tabes is considered.

Figs. 3 and 4 show how disparate is the involvement of the fasciculi composing the radicular nerve. Whether this is due to an elective affinity of a toxin for a particular sensory system, or whether it is due to an accidental incidence depending on the local differences of arrangement, cannot be affirmed with certainty. Clinical evidence seems to indicate that some of the fibers subserving the sensation of cold are spared relatively long; but no accurate data yet exist to indicate whether these fibers belong to the protopathic or epicritic systems demarcated by Head.⁶

The fibers subserving the sense of attitudes and those of deep pain are considered to be particularly early affected; and Marinesco⁷ has shown that the former is generally associated with the appreciation of vibration, which is much impaired in the course of tabes.

THE DISSOCIATION OF THE SENSIBILITY; THE CAUSES OF THIS.—Head has shown that these are two functions of deep fibers accompanying motor nerves. Spacing sense, thought by him to be in part a function of the same system, has not yet been sufficiently investigated in tabetics; but the implication of the two foregoing shows that the lesion must be one of the peripheral sensory neurons; for these two modalities of sensation become dissociated in the spinal cord, and would require two separate lesions for their implication, sense of position and movement passing homolaterally in the posterior column, while deep pain joins superficial pain to pass heterolaterally in a portion of Gowers' tract (Page May⁸). Touch and deep pressure impulses are, on the other hand, amalgamated into a single path as soon as they reach their relay neuron in the spinal cord. They mount cephalad in a heterolateral tract in the neighborhood of the ventral horn. Thus, if a lesion situated in the cord prevents the passage of impulses of deep pressure, it necessarily prevents the passage of tactile cutaneous impulses also, whether these are epicritic or protopathic, these distinctions being solely peripheral as regards their topographical separation (Head and Thompson⁹).

The disparate character of the sensory troubles is shown in "tabes supérieure" and by such cases of radicular astereognosis as were described by Déjérine¹⁰ and Noica and Averamescu,¹¹ of Bucarest,

⁶ The Consequence of Injury to the Peripheral Nerve in Man, Brain, 1905, xxviii, 116.

⁷ Semaine méd., 1905, xxv, 505. Rev. neur., 1907.

⁸ The Afferent Path, Brain, 1906.

⁹ The Grouping of Afferent Impulses within the Cord and Brain, 1905.

¹⁰ Rev. neur., 1904.

¹¹ Ibid., 1906, p. 99.

in which the palmar side and corresponding fingers could not identify the bodies palpated.

As other examples of such irregular incidence, the case cited by Dupré and Camus¹² and Camus and Sezary¹³ may be cited. A most demonstrative case was that of Babinski and Nageotte,¹⁴ in which the patellar reflex had persisted until death. Postmortem, it was found that the middle root zones were much less affected than the posterior root zones, while the reflex collaterals and the vertical fasciculus of the posterior horn were relatively conserved. This greater implication of the long fibers is, according to Nageotte, rather the exception. He bases this opinion principally upon the marked involvement of the external bandalette of Pierret, which he believes to be a tract of short fibers going to the column of Clark and not furnishing fibers to the column of Goll at all. These he believes to pass in the posterior external field, and perhaps the lower ones in Flechsig's oval. Raymond's¹⁵ case, too, is striking in this regard, as all deep reflexes were retained. Although hypotonia usually accompanies the lost tendon reflexes, yet even these are sometimes dissociated, as in this case. It would appear, too, that the hypertonic muscles of the tabetic may become even spastic, either by involvement of the pyramidal tract, as in so-called *tabes combinée* (Noica¹⁶) or by the development of paralysis agitans, as in the case of Bychowski.¹⁷ Reflexes too, may persist for some time although there is grave disorganization of afferent fibers from muscle, tendon, and joint, as evidenced by great muscular incoördination and lost sense of attitudes.

It is a pity that the two cases reported by Long¹⁸ were not examined as regards the vestibular nerve; for the preservation of the patellar reflexes was there combined with great ataxia; and yet postmortem, very little change was found in the lower spinal cord or roots.

The beautiful description by Gowers¹⁹ of the varying character of the lightning pains well shows the irregularity with which the fibers are attacked in the posterior roots; though his interpretation is inconsistent with the pathology for which this paper contends; for instance. "The superficial pains seem to be on the surface or just beneath the surface. They are usually felt at one spot, often only a few inches in extent, sometimes larger, extending down part of a limb, on the ears, on the face and especially about the mouth." "The pain is usually extremely brief, a stab or flash of pain, gone as soon as felt, but recurring." "These superficial pains have one remarkable effect; they make the skin tender. After they have

¹² *Rev. neur.*, 1902.

¹³ *Soc. de Biol de Paris*, 1905.

¹⁴ *L'Etat des reflexes tendineux dans le tabes compliquée de hémiplegie*, *Jour. de neur.*, 1907.

¹⁵ *Neurol. Zentralb.*, 1904, p. 786.

¹⁶ *The Pains of Tabes*, *Brit. Med. Jour.*, 1905, i, 1.

¹⁷ *Ibid.*, 1906, p. 1174.

¹⁸ *Rev. neur.*, 1906, p. 1182.

¹⁹ *Congrès de Genève*, 1907.

occurred a few times, sometimes almost as soon as they begin; the skin in that area becomes so tender that if touched the patient feels as if the place was an open sore. If the finger is drawn gently over it, the pain produced is often hardly bearable. A remarkable fact is that this occurs where sensibility to pain is lost." This description concords with that of the destruction or irritation of protopathic sensation: "the prick of a pin may occasion no pain whatever" (Head). Of the deep pains, the radicular character is as manifest; *e.g.*, "Pain after pain occurs at the same part, separated by a short interval." It is sometimes described as "like the pain of cramp when there is no muscular contraction." Of the girdle sensation, Gowers says that when superficial it is often accompanied by hyperesthesia, but when deep is without corresponding changes in the skin. Gowers also points out its occasional unilateral character.

The paresthesiæ are an endless list, and false interpretations are very probably due to the different degree of involvement of deep and epicritic fibers. His case of tabetic neuralgia without loss of the knee-jerks is a striking confirmation of this point of view; for the pains were entirely superficial; he could not bear the prick of a pin, but the sensation to touch was perfect. What is this but a protopathic paresthesia? There were five similar cases, all thus characteristic of root pain. Now, reference to Head's cases and those described by Lebar²⁰ will show that hyperesthesia of this nature is not due, as Gowers²¹ thinks, to irritation in periphery, but in nerve trunk. The nutritional changes also confirm Nageotte's point of view.

In one case a cutaneous erythema with numerous small white spots accompanied the pain;²² in one, the localized pain of the scalp was followed after a week by bending and breaking of the hairs about an eighth of an inch from their emergence. These phenomena conform to the trophic ones which Head²³ considers as dependent upon protopathic fibers.

The radicular distribution of the trophic changes is not consistent with their peripheral origin. The resemblance, too, of the arthropathy and osteopathy to those of syringomyelia is striking; but in the latter case the lesion affects the vasor centres in the intermediolateral column, and possibly certain other root fibers also; whereas, in the tabetic process it is the conducting fibers alone which are involved. The effect, however, does not differ. That these dystrophies are dependent upon the interruption of afferent impulses is, however, shown by the disappearance of the perforating ulcers when rest to the limb is enforced for a few weeks. It is only then that the ulcer can be adequately protected from the unfelt traumas which interfere

²⁰ Thèse de Paris, 1906.

²¹ On the Nature of Tabes, Brit. Med. Jour., 1905, p. 57.

²² The Dystrophies in Tabes, Brit. Med. Jour., 1906, p. 1267.

²³ The Afferent Nervous System from a New Aspect, Brain, 1905.

with its healing. Such an exposed surface would normally be so painful that the patient would be compelled to rest; but the painless mal perforante offers no inducement to this.

One of the hitherto unrecognized paresthesiæ is pruritis. Its characters, as pointed out by Lamy²⁴ and Millian²⁵ strikingly exemplify the irregular incidence of the tabetic process; for though generally bilateral, it may be confined to one side, and it usually coincides with a segmental area presenting other symptoms such as hypo-esthesia, or visceral crises. It naturally occurs only during the earlier stages of the disease.

The researches of Sherrington²⁶ and others have shown that the motor nerves of the anatomist are also accompanied by sensory fibers. These fibers proceed from end organs within the substance of the muscles, known as the muscle spindles. These spindles are sensitive to the deformation they undergo upon contraction of the muscles. It is possible that they are more exposed to noxious meningeal influences than other fibers which preceed straight into the posterior root. This perhaps explains the frequency of their involvement in the meningeal inflammation which leads to tabes dorsalis; though unpublished researches of Ransome, kindly communicated to me by Dr. Donaldson of the Wistar Institute of Anatomy, show that in rats at least these fibers are well mixed with those of cutaneous sensation by the time they reach the spinal ganglion. (See also Ingbert²⁷). When the clinician investigates the loss of deep pain by pressing the ulnar nerve behind the elbow, pinching the Achilles tendon or faradizing the sural muscles, he is ascertaining the diminution of a function conveyed by these same nerves. Each of these symptoms is very common (90 per cent., Déjérine²⁸) quite early in the disease.

As a matter of fact, other fibers are not spared; and if the disease is not arrested or cured or ended by death, all forms of sensibility are destroyed. The disease, however, progresses very slowly in most cases, although some rapid cases have been mistaken for acute myelitis, just as fulminating phthisis has for croupous pneumonia. As in tuberculosis, the individual lesions tend toward arrest, while the process tends to progress, the rate varying with the individual and the circumstances.

THE MUSCULAR ATROPHIES.—This tendency to local arrest explains the infrequency of serious myopathies; for Nageotte has shown that the anterior roots often degenerate. The possibility of their regeneration has not been generally accepted. But these and other cases, as well as the regeneration of the posterior roots, definitely shown by Bickles²⁹ experiments should compel the most

²⁴ Soc. méd. des hôp. de Paris, 1015.

²⁵ *Ibid.*, 991.

²⁶ Jour. Physiol., 1894, xxv.

²⁷ Jour. comp. neurol., vol. xiii, No. 11.

²⁸ Semeiologie du système nerveux, Traité de Path. Gén. de Bouchard et Brissaud, 1904.

²⁹ Ueber das Verhalten des proximalsten (extramedullären und pialen) Teiles der hinteren Wurzeln bei Degeneration und Regeneration, Neurol. Centralb., 1907, xx.

recalcitrant to accept the evidence of Nageotte's preparations as regards the anterior roots.

Muscular atrophy, however, is much commoner in tabes than earlier writers allowed. Witness the cases of Déjérine,³⁰ Souques,³¹ Marie,³² Oddo,³³ Camp,³⁴ and Raymond.³⁵ Nageotte's case of Aran-Duchenne palsy in a syphilitic, ataxic in the evenings, in which the patellar reflexes had disappeared, but who showed photomotor reflex iridoplegia and lymphocytosis, and whose symptoms began with ulnar neuralgia, is undoubtedly a tabetic, though the muscular atrophy may of course be of independent causation.

The recollection of the pathological principle upon which this depends would have saved many from their undue skepticism in this regard. It is sufficiently well known that nerves possessing a neurilemma will regenerate when no longer shut off from their trophic centre.

WHY THE ANTERIOR ROOTS REGENERATE EFFECTIVELY.—The trophic centre of the anterior root is the pyramidal cells in the anterior horn. Upon the absorption of the meningeal exudation which has interrupted that influence, they push out new fibers, which Nageotte has figured, and these, finding no interruption, wend their way once more to the muscular fibers which they innervate.

WHY THE POSTERIOR ROOTS DO NOT REGENERATE.—In the posterior root on the other hand, the portion of the fiber distal from its trophic centre, the spinal ganglion, loses its neurilemma on entering the central nervous system. Having suffered degeneration through the meningeal exudation upon the posterior root, that is, between itself and its trophic centre, its want of neurilemma deprives it of the power to regenerate, even when the trophic centre regains access; for the regenerating fibers, as Bickles' experiments show, stop short at the point of reflection of the pia mater at Obersteiner's ring (Fig. 7). Hence from the effects of this lesion no recovery is possible; but permanent disability through muscular atrophy due to anterior root lesions occurs only when the inflammatory exudation has eventuated in cicatrization.

Although I am one of the last to minimize the importance of the psychic factor in the temporary abasia of the tabetic, yet I believe that the future will show that these are sometimes, in part at least, due to the evanescent peripheral neuron palsies induced in the manner described. Faure³⁶ has already shown that ataxia of the trunk muscles is the real cause of some cases of tabetic abasia which were supposed to be psychic.

³⁰ *Rev. neur.*, 1905, p. 1218.

³¹ *Atrophie musculaire dans le tabes*, *Rev. neur.*, 1907.

³² *Rev. neur.*, 1905, p. 146.

³³ *Muscular Atrophy in Tabes*, *Univ. Penna. Med. Bull.*, 1905.

³⁴ *L'Encéphale*, 1907, p. 225.

³⁵ *Congrès de Lille*, 1906.

³⁶ *Ibid.*, 1906, p. 587.

REFUTATION OF OBJECTIVES TO THESE VIEWS.—David Ferrier²⁷ has summarized the objections to these interpretations of the pathology of tabes dorsalis by stating that the meningitis postulated is not a constant phenomenon, being more frequently absent than present, and when existing, being more of a secondary thickening than an inflammatory process. These objections are completely refuted by the cytological data of lumbar puncture. That lymphocytosis signifies meningeal inflammation, no one disputes. The frequent postmortem findings of thickenings rather than inflammations is explained by the law of fibrosis of individual lesions in the history of granulomas, the process itself continuing in other parts.

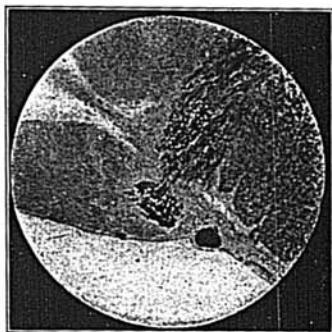


FIG. 7.—Showing the point at which the fibers of the radicular nerve lose their neurilemma. This is outside the cord. The degeneration shown is due to the passage of toxic lymph from a peripheral focus. (Marchi.) (From Orr and Rows.)

Ferrier further objects that in tabes well-marked intramedullary degeneration is often unaccompanied by neuritis or atrophy of the posterior roots. The experiments of Bickles effectively answer this objection, as previously indicated. Ferrier declares unintelligible what he terms the practical escape of the anterior roots, and thinks it contradicts the usual law of the greater involvement of the motor fibers in neuritis of mixed nerves.

This so-called law is an imagination of clinicians, as no one who has studied alcoholic neuritis can doubt; for in this affection of mixed nerves the motor fibers sometimes escape entirely. The fact that the motor neuron is involved in plumbism, often to the exclusion

²⁷ Brit. Med. Jour., 1906.

of the sensory, is due to its greater susceptibility to that particular toxin. Pharmacological studies show the existence of these selective powers. Ferrier, too, declares that it is extremely rare for meningitis to produce intraspinal degeneration by implication of the spinal roots. This statement is contradicted by the examination of cases of Potts' disease (Alquier,³⁹) spinal tumors, etc. Another argument he invokes is the segmental character of tabetic degeneration; but as segmental is a term in this connection convertible with radicular, the logic of this objection is not evident.

Another objection, its symmetrical character, does not accord with the facts, for Babinski⁴⁰ and Déjérine⁴¹ have shown that in the early stages a relative asymmetry is an important character in the tabetic process.

Another objection is the occurrence of degeneration elsewhere, as in cranial nerves, ciliary ganglion, and sympathetic system. The cranial nerve affections are, of course, due to a similar basal meningitis, while it is not established that the implication of the ciliary ganglion is an essential part of the tabetic process. The pathology of the sympathetic involvement will be considered farther on in this paper.

The fact that Thomas,⁴² in each of six cases, found decided changes in the spinal ganglion cells in no way invalidates the pathogenesis of Nageotte for "reaction à distance" is a well ascertained certainty. Besides, some of the cases of Redlich,⁴³ Marie and Leri⁴⁴ show distinct direct involvement of the spinal ganglia by an inflammatory process which has evidently extended from the meninges. This extension is not at all surprising when one remembers that even the purely mechanical lesions of the root described by Lejonne in cases of cerebral tumor sometimes reach the ganglia.

When the ganglion is directly affected by such an interstitial inflammation, it is not difficult to understand that the function of the cells becomes impaired, or even that their death may ensue. This will be manifested by a dentritic degeneration, which may well begin in the most distant parts, and hence give rise to the symptoms and postmortem appearance of the peripheral neuritis in tabes as described by Déjérine and others.

I do not, however, wish to exclude the primary peripheral origin of the nerve degeneration in some tabetics, for the infection at the root of tabes does not seem to be confined to the neural canal, as is shown by recent researches, which have shown the commonness of grave interstitial changes in the liver, spleen, and other viscera. Strong objection to the pathogenesis advocated in this paper has

³⁹ *Neuv. Icon. Salpet.*, 1907; *Gaz. des hôp.*, 1906, p. 687; 1906, p. 2.

⁴⁰ *Soc. méd. des hôp. de Paris*, November, 1901.

⁴¹ *Seméiologie du système nerveux*, *Traité de Path. Gén.* de Bouchard et Brissaud.

⁴² *Rev. neur.*, 1906, p. 573.

⁴³ *Arbeiten aus dem Institut f. Anatomie und Physiologie*, 1896, 985.

⁴⁴ *Traité de Méd.*, Bouchard-Brissaud, 1904, ix.

recently been made by Beutler⁴⁶ on account of the very marked vascular engorgement he found developing in the posterior columns of 14 cases most carefully examined. He believes this too great to be accounted for by the meningitis always present. But it is not unusual to find vascular engorgement of neural tissue undergoing degeneration, and, as previously pointed out, the meningeal lesions tend all the time to regress and leave little trace. As pointed out by Paviot⁴⁷ they are often destroyed in the postmortem manipulation.

Spiller, in a verbal communication, states that he invariably finds meningitis present in cases of tabes when carefully examined.

THE NATURE OF THE TABETIC PROCESS.—The transitions between frank tabes, general paralysis, and cerebrospinal syphilis are shown by such cases as the six of Guillan and Tahan⁴⁸ and the two of Camp,⁴⁹ in which decided meningitis of the cord and vascular syphilitic lesions accompanied the tabetic process. It is unfortunate that in the case of the latter the radicular nerves were not examined. It must not be forgotten that Nageotte and Babinski contend for the primary meningeal location of the disease, and they believe the lymphocytosis to be sufficiently presumptive of this. Marie and Guillain,⁴⁸ although they suppose the affection to be purely syphilitic, think that some peculiarity of the lymph circulation in the posterior columns confines the disease to that region. They do not attempt to explain the lesions on the posterior root, for Guillain's⁴⁹ experiments only show that the lymph circulation is from the pia toward the central canal.

The experiments of Orr and Rows further show a circulation of lymph from peripheral nerve via spinal ganglion as well as anterior root, into meninges and cord in its whole circumference. These and other considerations led Ford Robertson and Macrae⁵⁰ to interpret the tabetic process as a specific one due to a toxin, non-syphilitic, originating in diphtheroid organisms situated in mucous membrane, and whose activity was rendered possible by the lowering of bodily resistance due to antecedent depressing conditions, of which syphilis is probably the commonest.

That meningeal reactions may be produced in this way is shown by Orr and Rows, but as Robertson and Macrae are making further experiments to answer the bacteriological objections made to their contentions, the consideration of this theory must be left to a future occasion.

⁴⁶ Thèse de Lyon, 1905.

⁴⁷ Soc. méd. des Hôp. de Lyon, 1905.

⁴⁸ Soc. Biol., January, 1905.

⁴⁹ The Difficulty of Diagnosis between Cerebrospinal Syphilis, Univ. Penna. Med. Bull., xviii, p. 167.

⁵⁰ Rev. neur., January, 1903.

⁵¹ La circulation de la lymphe dans la moelle épinière, Rev. neur., December, 1899.

⁵² Rev. Neur. and Psy., 1904; May, 1905; February, March, and April, 1906. The Pathology of General Paralysis, Brit. Med. Jour., 1905.

THE SYPHILITIC NATURE OF THE PROCESS.—When the nature of the tabetic process is considered, it is more difficult to steer a clear course. Each in its way, the researches of Marie and Guillain, of Nageotte, of Orr and Rows, and of Ford Robertson and Macrae tend to show that the affection is one of lymphatic distribution, but here agreement ceases. For Nageotte, it is tertiary syphilis of the meninges, and the toxic lymph attacks the nerve root where it makes most intimate and long contact, that is, at the point where it receives its meningeal sheath.

The directly syphilitic nature of the process is supported by such facts as those of Brissaud and Oberthur,⁵¹ Dorlan,⁵² and of Duhot,⁵³ who reported two cases ensuing respectively two and four years after infection. In each of these the process was rapidly and definitely arrested by intensive mercurial treatment. Another significant fact is reported by Dalous⁵⁴ as follows: Out of 21 tabetics presenting pathognomonic, tertiary lesions, 9 denied knowledge of infection or recollection of secondary symptoms.

TABETIC SYMPTOMS IN SECONDARY SYPHILIS.—Symptoms regarded as pathognomonic of tabes are found even in the secondary stage of syphilis. Buttino⁵⁵ examined 70 syphilitics from this point of view, mostly during the first year of the disease. He found in a large number of cases a diminution of a transient nature in the light reflex. In 16 cases he found an evanescent leukocytosis. Babinski⁵⁶ found it in 7 out of 74 syphilitics of long standing without any nervous symptoms. Dufour⁵⁷ examined 611 patients, and found it only in 7, all of whom confessed syphilis. Charpentier⁵⁸ failed to find the sign in 1100 healthy individuals, while Sulzer⁵⁹ found a fleeting Argyll-Robertson sign in 12 out of 50 syphilitics he examined from this point of view. Dupuy Dutemps⁶⁰ and Cestan's research established the fact, and Mantoux⁶¹ collected 64 cases of this kind. Ravaut⁶² and Belètre found lymphocytoses in 58 out of 138 cases of secondary syphilis. This was often associated with exaggeration of the knee reflexes; and both conditions were recovered from under specific treatment. This exaggeration of the reflexes in no way modifies the point of view here sustained; for in polyneuritis, an exaggeration precedes a loss of the deep reflexes.

The association of lymphocytosis and reflex iridoplegia has been insisted upon by Babinski⁶³ and Nageotte, as has also the association

⁵¹ Congrès de Lille, 1906.

⁵² Thèse de Paris, 1906.

⁵³ Ann. de la Polyclin. Brux., 1903.

⁵⁴ Les lésions spécifiques dans le tabes, Rev. de Méd., 1904.

⁵⁵ Riv. di Pat. Nerv. e. Ment., 1906.

⁵⁶ Rev. neur., 1898.

⁵⁷ Soc. méd. des hôp., 1902.

⁵⁸ Thèse de Paris, 1899.

⁵⁹ Ann. de dermat. et de syph., 1901, p. 239.

⁶⁰ Le signe pupillaire d'Argyll-Robertson. Sa valeur seméiologique, ses relations avec la syphilis, Gaz. des hôp. de Paris, December, 1901, xxviii.

⁶¹ Thèse de Paris, 1904.

⁶² Soc. méd. des hôp. de Paris, 1901.

⁶³ Rev. neur., 1901; Soc. méd. des hôp., 1901.

of the latter with aortitis, now known as the syndrome of Babinski. Irregularity of the pupil was found 12 times in 80 old syphilitics, 5 of them without tabetic symptoms.

TABES NOT A TRUE SCLEROSIS.—The postulate of Weigert that the disappearance of the noble elements is followed by neuroglia proliferation has probably been utilized excessively in the interpretation of such scleroses as are found in locomotor ataxia; for in a pure secondary degeneration of the noble element of the posterior columns due to the excessive pressure of the cerebrospinal fluid upon the posterior roots in cases of cerebral tumor, Lejonne found no neuroglia proliferation in the 15 cases examined. Leri⁶⁴ proved by measurement also that the apparent increase of connective tissue elements in the corpus callosum in senile degeneration of the brain was in reality due merely to their closer approximation, from the disappearance of the nerve structures proper. In some cases the corpus callosum was less than half the usual thickness. The diminution of volume in the posterior column in tabes is a pathological fact; and the sclerosis is therefore not hyperplastic but cicatricial. The same considerations apply to the wasting of the posterior roots found by Phillippe⁶⁵ in advanced cases of tabes. This absence of sclerosis during an inflammation generally regarded as interstitial has been recently remarkably exemplified by Raymond⁶⁶ in a case of comparatively acute multiple sclerosis.

WHY THE SENSORY SYMPTOMS ARE IRREGULAR.—Why then is there irregularity and seeming anomaly in the sensory manifestations of tabetics? Is it not evident that in a process so widespread and irregular in its distribution, it must necessarily be of unequal incidence upon different portions of the nerve roots. That local peculiarities will determine the inclusion of now one bundle, now another, is well shown in such chronic involvement as Pott's disease, in which the research of Fry⁶⁷ shows that the same process may pick out and moreover affect unequally, here a band of protopathic fibers, there a band of epicritic; for in his case of cervical Pott's disease, the patches of anesthesia were most irregularly distributed in location, degree, and modality. These may be bundles of epicritic or of protopathic fibers.

One must take into account, too, the errors induced by the exceeding crudeness of the usual clinical investigation of the sensibility, before the researches of Head had indicated methods of precision.

The declaration of changes, more particularly in thermic sensibilities, antedating Head's research, is of small relative value, unless the actual temperatures employed are noted; while the fallacies of the clinical methods in the investigation of touch are by this time

⁶⁴ *Le cerveau senile*; Congrès de Lille, 1906.

⁶⁵ *Arch. de neur.*, 1897.

⁶⁶ *Jour. of Nerv. and Ment. Dis.*, 1907, p. 185.

⁶⁷ *L'Encéphale*, 1907, p. 225.

familiar to every neurologist. The conclusions of the careful psychometric research of Vaschide,⁶⁸ the only one of which I am aware, are vitiated by these considerations.

The investigation of the tactile sense by the haphi-esthésiomètre of Toulouse-Vaschide⁶⁹ is in reality one of the sense of deformation; for its principle is the appreciation of weight. This appears in the research itself, of which one of the conclusions is that the trichesthesia is less involved in the tabetic process. Now Head has shown that the trichesthesia is a tactile function entirely dissociated from the deep sensibility. Vaschide's research, therefore, furnishes valuable data when interpreted in the light we have obtained from Head. In his investigation of temperature, of which the amounts are given, the distinction of epicritic fibers is very manifest with regard to certain roots.

It is possible that the vulnerability of fibers even depends upon the distinction which has been attempted between those of slender structure myelinating late and subserving the life of relation, passing up the cord by the zones of Lissauer (which, however, Nageotte believes to be endogenous); and thick ones early surrounded by myelin whose function is that of internal tactility, including the conscious notion of attitudes subserved by the telencephalon, and the automatic tonus regulating sense presided over by the cerebellum. Bonnién⁷⁰ declares that the vestibular nerve is the homologue of the latter posterior fibers, and that a predilection of tabes for this nerve as against the cochlear portion, homologous with the finer fibers, is thus accounted for. As Head has shown, it appears that a single peripheral fiber may convey an impulse which in the cord may select either of these paths.

Were these latter functions always attacked concomitantly, it would be a conclusive proof that the process originated outside the central nervous system; for these two functions follow different paths in the spinal cord, the impulses which go to the cerebrum reaching there homolaterally via the posterior columns, and heterolaterally via the lemniscus, the optic thalamus, and the internal capsule; while the cerebellum receives its information homolaterally via the lateral columns, the restiform body, and the peduncle, and also heterolaterally via Gower's tract and in the main the superior peduncle.

The situation of these cerebellar tracts at the periphery of the cord causes a suspicion that they may be peculiarly susceptible to toxin by extension from the meninges, and may play a greater part in the ataxia of tabetics than one is willing at present to admit;

⁶⁸ *Bul. ins. gen. psy. de Paris*, 1903, iii, No. 5.

⁶⁹ *L'Examin. des Sujets* (dans le *Bibliothèque Internationale du Psychologie Expérimentale de Toulouse*).

⁷⁰ *Le tabes labyrinthique*, *Nouv. Icon. Salpet.*, 1899; *Le sens des attitudes*, Paris; *Le nerf labyrinthique*, *Nouv. Icon. Salpet.*, 1904; *Le Vertige*, 1904.

although recent researches of Crouzon⁷¹ and of Lejonne⁷² show how often they are affected in the mixed sclerosis of the aged. But clinical investigation has not yet ascertained whether these functions are in tabes always concomitantly effected. In the periphery, however, a single path appears to convey all impulses subserving sense of attitude whether these ultimately become conscious or not (Head).

VESTIBULAR TABES.—Pierre Bonnier lays great stress upon the vestibular causation of much of the ataxia as well as the oculomotor palsies of tabes. Though it cannot be said that his contention has been seriously examined by others in the light of careful clinical observation, yet it does not seem that clinical study, imperfect as it is, could have failed to distinguish between kinesthetic ataxia and that of vestibular origin. Nor is all disorientation of vestibular source.

This is proved even in the cephalic segment by the vertical stick illusion, in which, with eyes closed and head inclined, a grasped vertical rod appears to incline away from the side to which the head leans; and this phenomenon is quite unaffected by the condition of the semicircular canals or their nerves. It is probably due to kinesthetic interpretations coördinated by experience, and does not differ in the principle of its mechanism from such illusions as the up and down movements of the telegraph wires when passing them in the train, the appearance of the moon passing behind the clouds when in reality the clouds are crossing the moon, and from the deception of altitudes lent by distance, all these being due to the interpretation of unusual experiences by usual standards.

TABETIC STRABISMUS AND NYSTAGMUS.—Bonnier refers the oculomotor palsies to what he terms the bulbar syndrome provoked by irritation of the vestibular nerve, and irradiating via Deiter's nucleus toward one or more of the bulbopontine and mesencephalic nuclei from which Deiter's receives fibers, that is, those of the third, fourth, fifth, sixth, eighth, ninth, and tenth cranial nerves. He attributes to this mechanism the tinnitus, deafness, giddiness sudden falls, even without vertiginous sensations, which sometimes occur in tabetics, as well as Romberg's sign and the oculomotor palsies. He thinks that their basophobia is due to a similar overflow into the pneumogastric, producing a bulbar state corresponding to the emotion of terror, and due to what he terms an unconscious vertigo. He insists that we are no more conscious of the state of absence of vertigo than we are of the absence of thirst; and he can describe its numerous ways of occurring in only vague terms.

The fact that a patient often falls without any consciousness of vertigo is attributed by Bonnier to the suddenness by which Deiter's

⁷¹ Les scléroses combinées de la moelle, Thèse de Paris, 1904

⁷² Arch. gén. de méd., 1905, p. 3009.

neucleus is stimulated by peripheral irritation of the great posterior root, the eighth nerve. He falls before a nerve impulse reaches the cerebrum and constitutes an impression in consciousness. He compares this fall to that of the epileptic without aura.

Etienne⁷³ has recently reported a similar case.

It remains to be seen whether these cases really differ from those described by Gowers⁷⁴ as vagal attacks, which often appear to be of true epileptic nature. They, however, differ in that consciousness is retained in the latter.

THE OPTIC ATROPHY IS DUE TO BASAL MENINGITIS.—The pathological evidences derived from the examination of the optic nerve in tabetics strongly confirm this view of the pathogenesis of the tabetic process.

Originating in the large ganglion cells of the retina, these fibers run a long course in close contact with the meninges until they reach their destination in the superior colliculus and the lateral geniculate body; and although they are not the homologues of posterior root fibers, but of the arcuate fibers in the medulla and the medial lemniscus in the midbrain, yet they are subjected on account of their situation to the same extension of inflammatory processes upon the meninges as are the posterior roots. Although it has been sustained by Popoff and Von Grosz⁷⁵ that the lesions were primitively retinal, on account of the disappearance of the ganglion cells of that membrane, yet the persistence of many cells there, even when the optic nerve is completely atrophied, has been shown by Marie and Leri.⁷⁶ This would seem to indicate that the process is not primitively ganglionic. It is right to say, however, that Dupuy-Dutemps⁷⁷ strongly contends that the real cause of the lost light reflex is a diseased condition of the nerves of the iris.

The amaurosis proceeding from the periphery, and the optic atrophy are the clinical concomitants of this peripheric invasion of the optic nerve. The rarity of the commencement of a tabetic amaurosis by a central scotoma is only to be expected when one recollects that the macular bundle runs in the centre of the optic nerve except at the very beginning of its course, where it is peripheral, upon the mesial side of the nerve (Henschen⁷⁸). It is its involvement at this spot which accounts for the few cases of central scotoma of tabetic causation (Galezowski⁷⁹).

The central scotoma which ensues upon a hemianopic contraction of the visual field has, however, a different significance; for this is due to the invasion of the macular bundle in the heart of

⁷³ Un cas de syndrome de Bonnier, *Rev. neur.*, 1907, p. 1025.

⁷⁴ Vagal Attacks, *Lancet*, June, 1907.

⁷⁵ *Congrès Internat. d'Ophthal.* Utrich, 1889.

⁷⁶ *Nouv. Icon. Salpet.*, 1904, p. 368.

⁷⁷ *Con. d'Ophtal. de Paris*, 1906; *Annals Oculist*, 1905.

⁷⁸ *Klinische und Anatomische Beiträge zur Path. des Gehirns*, Zweite Theil, Upsala, 1892.

⁷⁹ *Le fond de l'œil dans les affections du système nerveux*, Thèse de Paris, 1904.

the nerve in the course of an infiltration proceeding usually from the mesial side (R. Jocqs⁸⁰) (Galezowski).

The very bad prognosis in these cases is due to the smallness of the macular bundle and the fact that a hemiopic contraction is a proof that this bundle has been reached by the inflammatory process. The bundle in the bandalette described by Marie and Leri,⁸¹ which they have always found intact in common with that of Meynert⁸² in cases even of complete optic atrophy, has nothing to do with the optic fibers, but runs between the sublenticular region and the basal optic ganglion of Meynert.

Nageotte believes that the zonula of Zinn is the usual site of the lesion.

THE SYMPATHETIC SYMPTOMS ALSO ARISE RADICULARLY.—It may appear strange that our point of view should be confirmed by those tabetic symptoms which are usually referred to the sympathetic nervous system. It will be recollected that Duchenne at one time thought that the lesions found in the cord and roots might be due to a hyperemia induced by a paralysis of the great sympathetic by the tabetic process, and a memorable controversy arose with Vulpian with regard to this. The latter, indeed, found sympathetic lesions only exceptionally, while Charcot,⁸³ in 1888, stated that even in subjects who had suffered for years from gastric crises, there was no change in the nerves and ganglion of the solar plexus. Raymond,⁸⁴ in 1894, thought the integrity of the sympathetic was demonstrated.

This was the state of matters when Jean Ch. Roux⁸⁵ was inspired in 1900 by Déjérine to investigate the question. He examined seven tabetics and ten subjects dead of other nervous and other diseases. In all the latter he found the sympathetic healthy, while in not one tabetic did he fail to find marked and identical modifications in the sympathetic chain and the great splanchnic.

In both of these and in all the cases he found that the small myelinated fibers were diminished in number, in the chain from 4400 to about 500, in the splanchnic from about 2000 to about 1150. It will be recollected that these myelinated fibers are derived from the spinal cord, certainly from the intermediolateral column, where Bruce⁸⁶ found as many as 8000 characteristic cells, and possibly from other regions of the gray matter. They are slender fibers possessing a myelin sheath. They leave the cord by the anterior and posterior roots; and they terminate around the sympathetic ganglion cells, from which another fiber takes up the nerve impulse

⁸⁰ Atrophie optic tabétique et scotome centrale, Extrait du Recueil d'Ophthalmologie, April, 1906.

⁸¹ Rev. neur., 1905, p. 193.

⁸² Leçons cliniques, 1888.

⁸³ Thèse de Paris, 1900.

⁸⁴ The Intermedio-Lateral Tract., Rev. neur. and Psy., 1907.

⁸⁵ Psychiatry, Trans. by Sachs, 1887.

⁸⁶ Leçons cliniques, 1894.

to terminate in muscle and gland. These latter are the so-called fibers of Remak, and possess no myelin sheath. Neither communicate with the posterior root ganglia.

Now, unless Roux's findings are due to an autonomous lesion, they must be secondary to a process in one or both of two regions, that is, the gray substance of the cord or the spinal roots; and as lesions of the gray substance of the cord have scarcely been observed in tabes, this degeneration of the myelinated sympathetic fibers must be due to the root lesions Nageotte has described. That this is the case Roux showed by observing their degeneration in cats after section of the spinal roots.

That this is not a general part of a peripheral neural degeneration was shown by the conservation of other peripheral nerves such as the intercostal. The sympathetic ganglia, too, were not diseased, nor are any changes presented by the large myelinated fibers which come from the spinal ganglia, as Roux shows these latter were intact in all the cases. Moreover, the diminution of slender myelinated fibers was proportional in the sympathetic to their decrease in the posterior roots in each case.

It is to the destruction of these that Roux attributes the anesthesia of the testicle, bladder, breasts, and trachea, as well as that of the stomach, which he has specially studied. The lack of the sensory innervation is for him the cause of the vesical hypotonia with its consequent ataxia, to which is due the micturitional difficulties of the tabetic. It is to a similar mechanism he attributes the gastric atony, which he believes responsible for much of the ill health and even of some of the gastric crises occurring in this disease.

Genouville⁸⁷ showed that when a tabetic bladder is touched by a sound a desire to urinate often follows, while this is not the case in normal persons. To a paresthesia of similar mechanism, Roux attributes the gastric crises and segmental referred pain which his observations show to be frequently provoked by dietetic errors or by drugs. The mechanism does not appear to me to differ in principle from that in a case I now have under observation, in which athetoid movements of the toes take place on stimulation of the skin even by exposure to cold.

The researches of Köster⁸⁸ into the trophic changes ensuing upon experimental section of the posterior roots show them to differ in no way from those in tabetics and thus confirm this hypothesis.

I may then conclude:

1. Tabes dorsalis is a secondary degeneration in the posterior columns, due to a chronic meningitis, very probably of syphilitic nature.

⁸⁷ Thèse de Paris, 1894.

⁸⁸ Zur. Phys. der Spinalganglion und der trophisch Nerv. and zur path. d. tabes; Leipzig, Engelmann, 1904.

2. The arrangement of the meninges surrounding the radicular nerve renders it peculiarly susceptible at that spot to mechanical or toxic injury.

3. The unequal incidence of the affection upon different fibers of the posterior root is probably due to unascertained peculiarity of structure or arrangement of fasciculi, rather than to any selective toxic influence.

4. The lesions tend toward resolution and arrest, even though the process may continue during the life of the individual.

5. With this arrest, regeneration tends to occur in the radicular nerve, the amount in the anterior root being relatively considerable while that in the posterior root is less in amount and functionally insignificant, as a rule.

6. The otherwise inexplicable vasomotor and cranial nerve symptoms and postmortem findings in this disease are shown thus to be necessary concomitants of the tabetic process.

7. The question of the pathogenesis of the polyneuritic manifestations found in tabetics is not yet answered.

The practical appreciation of the foregoing considerations to the treatment of tabetics need not be enlarged upon, as it does not come within the scope of the title of this paper; but when correctly applied, I believe another illustration will be afforded of the principle, *Naturam morborum curationes ostendunt*.

A FURTHER CONTRIBUTION TO THE HERPETIC INFLAMMATIONS OF THE GENICULATE GANGLION.

A SYNDROME CHARACTERIZED BY HERPES ZOSTER OTICUS, FACIALIS, OR OCCIPITOCOLLARIS, WITH FACIAL PALSY AND AUDITORY SYMPTOMS.

By J. RAMSAY HUNT, M.D.,

CHIEF OF THE CLINIC AND INSTRUCTOR IN NERVOUS DISEASES IN THE CORNELL UNIVERSITY MEDICAL SCHOOL, NEW YORK; NEUROLOGIST TO THE CITY HOSPITAL; CONSULTING NEUROLOGIST TO THE BABIES' HOSPITAL, AND THE NEW YORK EYE AND EAR INFIRMARY.

THIS communication is a further elaboration of a syndrome presented at the American Neurological Association, in June, 1906.¹ At that time, three well-recognized clinical types of herpes zoster of the face and neck, with facial palsy and acoustic symptoms, were fused into a single group. The characteristic symptoms

¹ Herpetic Inflammations of the Geniculate Ganglion: A New Syndrome and its Complications, *Trans. Amer. Neur. Assoc.*, 1906; *Jour. of Nerv. and Ment. Dis.*, February, 1907; *Archives of Otolaryngology*, August, 1907.